ally giving an accurate and definite statement of the occupation of the deceased. This is a matter of real importance, and a little attention on the part of physicians will make possible a much more valuable report.

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The Mental Hygiene War Work Committee of the National Committee for Mental Hygiene is anxious to obtain the names of psychiatrists and neurologists who are willing to give part-time service in the examination of National Guard troops in their vicinity. The recent decision of the War Department to examine the National Guard troops in their armories before sending them to camp, makes it necessary to secure at once a large number of examining physicians. To meet the situation the Surgeon General of the Army has arranged to accept for this work qualified physicians on contract. A physician may contract for specified duty, at a specified place, for a specified time, or for part time. This latter provision makes it possible for many physicians who cannot take out commissions, or who cannot give all of their time to the work for a period of months, to give part-time each week. Further information can be received from Dr. Frankwood E. Williams, 50 Union Square, New York City.

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Do not pay money to unauthorized agents who claim to be peddling state medical directories. Please report to us at once any such solicitation.

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There is probably no class of dependents whose welfare has been more completely neglected, who have received less scientific study and care, than the aged. The child dependent has the world for its guardian; the aged dependent is disowned by its own. There are scores of works dealing with the child in the home and in institutions; until recently there was not a single work considering the institutional care of the aged, not a journal of any kind sufficiently interested in the welfare of the aged to devote special space to this subject.

The keynote of the treatment of aged persons is mental stimulation, to overcome the mental depression natural to the aged, especially those who are dependent upon others for their support. This mental stimulation may be brought about through recreation or amusements, or through arousing an interest in the affairs of the day, or in agreeable work, or in a hobby, or in self or another, or in the institution itself. I saw this well exemplified in a home for aged pensioners near Vienna which I visited a few years ago. The inmates were proud of their institution, and my guide took pains to show me how they helped each other to keep their dormitories, dining rooms and other rooms, halls, and walks clean and neat. They were proud of the appearance of the shops and of the skill of the inmates who worked there. The men took pride in their appearance, and before going out they washed themselves and brushed their clothes, hats and shoes. They had a band and an orchestra composed of inmates who gave occasional performances and always had appreciative audiences. Provision was made for their recreation; there was a well stocked library, and a canteen was established for them on the grounds. The canteen was maintained from the proceeds of knick-knacks made by the inmates, of concerts by the band, contributions from visitors and a slight profit on the sale of things supplied by the canteen, all of which went into a common fund. Similar provision for the recreation of the inmates could be made in all homes for the aged at but little cost.—I. L. Nascher. M. D., New York, Modern Hospital, July, 1917.

# Original Articles

## SPLENECTOMY IN PERNICIOUS ANEMIA.

By HARRY M. SHERMAN, M. D.,
F. A. C. S., San Francisco.

"The removal of a spleen damaged by rupture, torsion of its pedicle, or loosened from its position in the abdominal cavity, is an operation in itself not difficult and unlikely to lead to any untoward consequences" (Thursfield and Gow). Therefore the spleen itself may be assumed to be an organ not wholly essential to the life of the individual, and "that its functions, whatever these may be, are capable of performance by other tissues in the body." Sir John Bland-Sutton, in the British Journal of Surgery, Vol. I, No. 2, published in October, 1913, quotes Pliny (A. D. 23-79) as saying of the spleen that "sometimes it is a peculiar hindrance to runners so that they burn it away from those runners who are incommoded by it," and points out that the traditions before the Christian era showed that men and animals could live without a spleen. In 1581 (Bland-Sutton; Adelman), Viard tied the vessels of and removed a spleen that had prolapsed through a wound involving the false ribs, and the patient recovered. Nearly 300 years passed, however, before physiology took up the subject, when, in 1841, Bardeleben extirpated the spleen and thyroid of a dog and the animal lived six years in health afterward.

Vulpius in 1894 (Bland-Sutton) noted that in young animals who had abundant red marrow, splenectomy could be done with no sequent bad effects, but in older animals there was always fever and emaciation. Moreover, Vulpius noted in one of his splenectomized dogs enlargement of the abdominal lymph glands and a number of miliary bodies in the peritoneum and omentum which structurally resembled the Malpighian bodies of the spleen. That they came as a result of the splenectomy is rendered doubtful by a case report of Albrecht (Bland-Sutton) who saw in a young man dead of nephritis, a spleen only the size of a walnut in the usual place, while sprinkled throughout the abdomen—on the ligaments of the liver, on the mesentery and mesocolon and on the peritoneum generally—were a multitude of little spleens, each of which was red, the pulp histologically splenic in character, and each having a capsule and a covering of peritoneum.

In spite of the opportunities of modern times for experimentation, physiologists have not yet been able to ascribe to the spleen a definite function. Thursfield and Gow say of its functions, *"whatever these may be,"* and Bland-Sutton speaks of it as an enormous lymph gland, the removal of which is likened to the removal of enlarged lymph nodes from the neck, axilla or groin.

The present case is reported chiefly because it illustrates the common or normal reaction of patients with pernicious anemia to a splenectomy, and it includes a necropsy report which is practically classical.

The patient, a man 51 years old, was sent to

me by Doctor W. C. Chilson of Tulare. He was an apiarist. He had had indigestion off and on for 15 years. When he had the indigestion he would vomit and so get relief. Sometimes he vomited blood which he thought came from his throat. The last hematemesis was 5-6 weeks before coming to me. He was anorexic and constipated, the stools sometimes being clay colored. He slept badly. He had lost 12-15 lbs. of weight.

Examination in the hospital showed emaciation and jaundice. Clinically the abdomen was negative; the urine was normal except for a trace of albumen; the stool was negative. The blood showed 27% hemoglobin, 850,000 red cells, 5400 white cells. The red cells were irregular in shape and size with poor coloring. There were megaloblasts and a few megalocytes. The white differential count was: polymorphonuclears 79%, large mononuclears 2, small mononuclears 19. There were no malarial parasites. The Wassermann reaction was negative. After a test meal the stomach contents showed no free hydrochloric acid, no combined hydrochloric acid, no occult blood, no lactic acid, no Boas-Oppler bacilli.

Doctor Harold Hill and Doctor G. H. Evans saw him with me and agreed in the diagnosis of pernicious anemia. Under dietetic and arsenic treatment he improved and went home, where he continued the arsenic, but ate, I am quite sure, in a somewhat erratic way, being a man much given to the having of ideas, and once he became possessed of an idea he was wholly dominated by it. For instance, he was unalterably convinced that his condition was due to the inhalation of formic acid in working over his bees.

Five months later he returned and was found to have relapsed, the hemoglobin was 30%, 1,450,000 red cells. Color index 1.+, irregularities of shape and size, many megalocytes, no nucleated cells, no malarial parasites. The white cells were 3400.

A temporary improvement followed rest and food and then he again failed, the red count going down to 950,000, still irregular in shape and size with megalocytes, microcytes, nucleated cells, and poikilocytes. The blood pressure was 110 mm. Hg. At this time he complained of parasthesias of hands and feet, he was very weak physically and somewhat demented.

Two days later he was transfused from his son; the blood flowed for 20 minutes and during this time the patient's hemoglobin rose from 12% to 25%, and his pulse fell from 112 to 92. Later in that day his hemoglobin reached 35 and there were 2,050,000 red cells. The next day the hemoglobin was 40 and the red cells 2,200,000.

Two days after the first transfusion I did a second from another son and so ran his hemoglobin up to 53 and his red cell count to 2,800,000.

The next day I removed the spleen. The organ was not enlarged, there were no adhesions, the artery was tied before the veins, silk was used, no difficulty was encountered. Nothing was palpably wrong in the other abdominal organs.

From this operation the patient made a normal recovery. The parasthesia in the hands and feet lessened, more in the hands. The hemoglobin continued to rise and three weeks after the splenectomy it was 70, the red cells remained 2,800,000.

Two months later the hemoglobin was 92. Red cell count 3,500,000, numerous megalocytes, occasionally a red cell contained one, sometimes two, small colloid bodies taking the basophilic stain by Wright's method, the so-called Howell-Jolly bodies. The white count was 8,200.

This count in the end of January, 1915, three months after the splenectomy, was the high tide of the improvement. From that time all counts showed him to be failing. His blood picture showed this, and his conduct in all particulars did the same. He complained especially of the parasthesia which reached from his hands to his upper arms and from his feet to his waist, and as a phase, I think, of this he told of a dragging in his abdomen.

After going down hill six months he had hemoglobin 45%, red cell count 1,400,000, many megaloblasts and megalocytes and a few poikilocytes.

In September he returned very weak, dyspnoeic on slight exertion. The parasthesia was his chief complaint: it included the hands to the wrist, and the feet and legs, etc., up to the waist. He again complained of the dragging inside.

His hemoglobin was 20%, red cells 1,000,000, white 4,000, the reds showed the same characteristics as before, but no Howell-Jolly bodies were found. Purely as a temporary measure I transfused him again from his son. The father could live for a while on the son's blood, and the son was wholly willing to give it. By this we ran the father's hemoglobin up to 30, the parasthesia left the hands and only extended up so far as the knees from the feet. The improvement was not as much as I had hoped for, but the blood clotted in the tube after 20 minutes of running and so stopped the transfusion and I did not think it fair to the son to make another connection. The gain lasted only a short time and then he began to fail rapidly. But he was anxious to live and returned in November with two daughters to have more transfusions, but these I advised against, though I did have the hemolysis possibilities studied.

His hemoglobin was but 10, his red cells 500,000, they were very irregular in size and shape. No nucleated cells seen. The white cells were 3,000.

He died about 13 months after the splenectomy.

Thursfield and Gow, St. Bartholomew's Hospital Reports, Vol. L, Part 1, Article, Splenomegaly —Splenectomy, quote the fact that the spleen is not an organ essential to life, and Noguchi's blood studies on one man who had lost his spleen —not a pernicious anemic—in whom he noted first a "diminution in the total number of the polymorphonuclears . . . . and increase in the absolute numbers of the lymphocytes . . . and that the eosinophiles are increased; and that later

still the blood picture resumes a perfectly normal state." They quote and experience variations from these studies, and finally conclude that the effect on the blood varies in different individuals and instances; usually there is a quick increase in both reds and whites with a gradual return to near the norm, but with tendencies in the direction pointed out by Noguchi.

In pernicious anemia they quote Eppinger, Klemperer and Hirschfeld on the indication for splenectomy. "Eppinger's view may be stated as follows: The iodine number of the blood fat varies: it is at a minimum after experimental splenectomy; it is at a maximum after poisoning with toluylene diamine. In human beings high iodine numbers are found in all hemolytic processes. Secondly, the normal urobilin content of the stools is about 0.15 grm. per diem. In hemolytic diseases it is enormously increased, to 3 or 4 grm. per diem. In hemolytic icterus the effect of splenectomy is to lower both the output of urobilin and the iodine number of the blood fats. Eppinger therefore believes that splenectomy is indicated in all patients in whom these phenomena are present, holding that they provide an index of the morbid hemolytic influence of the spleen."

Klemperer and Hirschfeld, on the other hand, believe that removal of the spleen provides a stimulus to the hemopoietic functions of the bone marrow, and that the normal function of the organ is to regulate the production of the erythrocytes. Its removal, they point out, tends often to the appearance of a polycythemia.

Eppinger therefore can hope for a permanent cure of the disease; Klemperer would expect only an improvement in the blood condition. Hirschfeld states plainly that splenectomy is only a symptom remedy.

Thursfield and Gow refer to 21 cases of splenectomy in pernicious anemia which they have traced. They quote no absolute cures. They point out the long periods of improvement and that in the exacerbations treatment will often inaugurate a remission; when that does not follow it may be inferred that the patient is entering the final phase, and splenectomy may offer a release from this, inaugurate another remission and so prolong life and even an active life for a limited time at any rate.

Dr. H. C. Moffitt in *The American Journal of the Medical Sciences,* December, 1914, quotes the same authorities and to the same effect. Eppinger bases the indications for splenectomy on increased urobilin in urine and stools as an index of pathologic hemolysis, and "has pointed out that the iodin content of the blood after the removal of cholesterin and cholesterin esters runs fairly parallel to the degree of pathologic hemolysis. Eppinger reported five splenectomized patients after times varying from a few days to nine months, in all of whom there were icteric coloring and large quantities of urobilinogen in the stools. In these cases splenectomy stopped the blood destruction." Moffitt saw one of these patients six months after the operation, and while he had gained weight and had returned to business, his color was not normal and his blood was still of the megalocytic type.

Of Klemperer and Hirschfeld, Moffitt says, "the theory is advanced of some normal regulating function of the spleen upon the activity of the bone marrow; after the removal of the spleen, particularly when diseased, this inhibition is released and normal and abnormal erythrocytes are thrown rapidly into the circulation. In nine cases there were great numbers of normoblasts and erythrocytes with Howell-Jolly bodies in the peripheral blood. In one case seven months after the operation megalocytes were prominent in the blood picture and there was no difficulty in recognizing the pernicious type. The authors agree that splenectomy may bring about a remission when other means have failed, but Hirschfeld states plainly that 'splenectomy is only a symptom remedy.'"

In all Moffitt records thirty-three cases with full reports of clinical and laboratory findings. Of these, eight died immediately or soon after operation. Of the remaining twenty-three many improved rapidly in most all particulars, but none lost their pathologic blood picture, and while many of their symptoms were ameliorated they were none of them restored to health, for the most that he can say for the operation is that "cases are reported apparently cured after periods of three to nine months (Mosse, Eppinger); but the time is much too short to permit any such statement.

Moffitt argues that splenectomy may be counted on with fair hope to bring about a remission when other means have failed.

There have been a number of these cases reported in the past year but the time forbids their being drawn into this paper. I may merely cite Percy's report of five patients in whom he used the technic I had tried, viz., massive transfusion and then the splenectomy. One of these he cites as being satisfactory, and the other had been too recently operated upon to permit any deductions.

In the case of my report the result certainly showed that the splenectomy had but a temporary effect. That it did have some effect I am sure, for the patient was failing though under active treatment and so can be assumed to have been in the terminal phase when splenectomized. He had two massive transfusions, but their effect alone, at the best, could not have been expected to have lasted more than three weeks, while the patient gained steadily for three months after the splenectomy and did not get into a desperate condition for five months more, so that the method and the operation seem to me to have been to some extent justified.

Mr. H. G. Brown: Died November 28, 1915; autopsy November 28, 1915.

Man about 50 years old, poorly nourished, emaciated, skin dry, light brown hue, no edema, no jaundice. Skin and visible mucous membranes very pale, superficial lymph glands not enlarged. Hair partly gray, pupils equal, not dilated, nose normal. Teeth good, well preserved and kept,

tongue pale, coated, scars from transfusion operations at both elbows. Long white scar on abdomen in left mammary line from costal border to below level of umbilicus. Abdomen flat. External genitals normal. Thin layer of submucous fat, bright yellow color. Muscles very pale and dry, have a waxy appearance, rigor fairly well marked, beginning calcification of costal cartilages. Abdominal viscera: flexure and adjoining parts of transverse and descending colon bound to under-surface of abdominal scar near the upper third by long, quite firm adhesions. Omentum drawn up and attached firmly in the same place. No other adhesions in the abdomen. Spleen absent. No adhesions in or near the splenic region. No fluid in abdomen. Peritoneum dry. All viscera very pale. Right kidney normal size. Capsule very slightly adherent. Surface smooth, cut surface very pale with a yellowish tint. Markings normal, ureter normal. Right suprarenal normal. Left kidney and left suprarenal same as right. Bladder contains small amount of pale, cloudy urine, mucous membrane pale. Seminal vesicle full of albuminous thick fluid, prostate apparently normal. Urethra normal. Sigmoid and rectum empty. Adherent thick mucous to the pale mucous membrane. Colon empty except for small, hard masses of material resembling charcoal. Cecum full of bile-stained liquid. Appendix small and atrophic. No adhesions. Ileum, jejunum and duodenum empty except for small amount of bile-stained mucous. Small mucous membrane in the lower ileum, very thin. In this region the muscle wall is also very thin. Stomach small, contains small amount of thick mucous. Stomach mucosa thin, smooth and very pale. No hemorrhages anywhere seen, no ulcers. Bile duct open. No parasites found. Pancreas very pale, otherwise normal. Liver at margins of ribs, normal size, surface smooth. Cut surface very pale, centers of lobules stand out plainly as though pigmented. Diaphragm: 5th rib right, 6th rib left. Right pleural cavity almost entirely obliterated by old form of adhesions. Right lung, old scar at apex, the whole lung very edematous. Bronchioles filled with frothy liquid, very numerous, very small bronchopneumonia patches. Left lung same as right, except no adhesions. About 300 cc. clear fluid in left pleural cavity. Heart: no fluid in pericardium, heart normal size. Small post-mortem clots in cavities, valves normal, right ventricle, 6 m.m.; left 11 m.m. Cut surface flabby. numerous small, light yellow, pin-point spots in the muscle wall especially well shown beneath the endocardium in wall of left ventricle. Aorta normal. Thoracic and abdominal vessels normal. No visible enlargement of any of the abdominal or thoracic lymph glands except the peribronchial lymph glands which are anthracotic. No gall-stones. Gall-bladder normal. No thymus remnant found. Thyroid small, posterior capsule very adherent to trachea. Cut surface very solid and dry. Bone marrow of rib very soft and pale red color. Spinal cord quite firm meninges; pale, otherwise normal. Cut surface of cord apparently normal markings.

Anatomical diagnosis: Pernicious anemia; atrophy of intestinal mucosa; brown atrophy of liver. Fatty degeneration of heart; old adhesive pleurisy on right; edema of lungs, with terminal bronchopneumonia.

**St. Luke's Pathologic Laboratory.**

Pathologic report Dceember 6, 1915.

Section of lung shows very marked edema.
Section of thyroid gland shows atrophy and fibrosis.
Section of heart muscle shows extensive fatty degeneration and beginning brown atrophy.
Section of prostate shows marked fibrosis.
Section of stomach shows atrophy of the mucous membrane.
Section of liver shows marked brown atrophy in the centers of the liver lobules. Much fat in the fine droplets about periphery of the lobules.
Section of gall-bladder shows mucous membrane atrophic.
Section of kidney shows numerous small scars in the cortex in which are collections of leukocytes. The glomeruli in these scars show hyaline degeneration of the capsule. The capillary loops are intact. Much interstitial fibrous tissue between the collecting tubules. Most of the tubules are filled with granular material.
Sections of spinal cord show no lesion.
Section of suprarenal capsule show no lesion.
Sections of aorta show normal structure.
Section of small intestine in the lower ilium shows atrophy of the mucous membrane.
All sections show severe anemia.
Smears of bone marrow show many nucleated red cells, some very large, some small and irregular.
Many myelocytes, few of which contain eosinophil granules.
Diagnosis: Pernicious anemia; fatty degeneration of the heart; atrophy of all organs; arteriosclerotic kidney.

**Discussion.**

Dr. H. C. Moffitt: I would like to say that I think it wrong to consider pernicious anemia as a disease of the blood. If we regard it so, we will miss a number of cases.

I saw an interesting man to-day and, queerly enough, he comes from near Tulare. His blood count was normal in October. From the nature of his paresthesia you would have to regard him as a man who would probably have pernicious anemia. To-day his red cells are not as large as we usually see, but are well above normal, and the blood picture is otherwise that of pernicious anemia. The paresthesia began in the typical way and jumped suddenly from ankle to knee, knee to waist. Paresthesias like this (apart from a few disseminated spinal cord lesions) outside of pernicious anemia are extremely rare.

The cases of disseminated spinal cord lesions described by Batten and Collier have, many of them, a terminal blood picture of pernicious anemia. Lesions in the spinal cord, stomach and intestines are quite as important as lesions in the bone marrow, and for this reason it seems to me wrong to talk of splenectomy as a possible cure for pernicious anemia. It will, as Dr. Sherman says, give us one method of bringing about remissions, but it is almost impossible to say when a patient with pernicious anemia will not spontaneously have remissions.

If we do advocate splenectomy, we must realize thoroughly that we are relieving one phase of the disease—the action of spleen on bone marrow, but are not at all reaching the fundamental cause of the disease.

Dr. P. H. Pierson: I have been interested in this subject, especially in the urobilin output, which was studied in a dozen cases on the services of Dr. Edsall and Dr. Cabot of the Massachusetts General Hospital. Dr. Robertson did the work by the Wilbur and Addis method, by extracting the urobilin from the 24-hour stool with acid alcohol, and diluting this extract until the characteristic spectroscopic absorption bands of urobilin disappeared (about 5000 dilution in normal individuals). In the pernicious anemia cases studied, they found the dilutions ran up as high as 16,000 to 46,000. The effect of salvarsan on this urobilin output was practically nihil. Transfusion seemed to increase temporarily the amount of urobilin output because of the stimulation of the bone marrow. After splenectomy the urobilin dropped in four out of five cases to practically normal. One went below normal to 3500. In one other case the amount had risen considerably a few months later, and that case was not doing well. In summing up his article, he advocates the use of urobilin estimation (which shows the amount of blood destruction) to indicate whether splenectomy is advisable. Cases with spinal cord manifestations were not splenectomized because of the probably unfavorable results that would take place.

Closing discussion, Dr. Sherman: There is very little for me to say. I do not think for a moment that splenectomy can be considered more than an inaugurator of remission when nothing else will do it. If the individual is going into the terminal

phase in spite of treatment, it is perfectly fair, it seems to me, to advocate it.

I will have to acknowledge that in this patient no test of the uroblin output was made nor of the fragility of the blood cells.

One point interested me, although I do not fully know its value, and that is the Howell-Jolly bodies in the blood. There were relatively few in this instance, and if their presence indicates, as it may, a speeding up of the blood-making function and the calling out of the young cells earlier than normal to fill the ranks in the vessels, their absence would possibly mean a failure at the very point of origin of the red cells—a failure to generate reds rather than a too rapid destruction of them.

## SOME UNUSUAL ASPECTS OF EXOPHTHALMIC GOITER.*

By GEORGE D. BARNETT, M. D., San Francisco.
From the Medical Division of the Stanford Medical School.

With the great increase in pathological and experimental work on exophthalmic goiter during the past few years the focus of attention has shifted somewhat from the field of diagnosis to that of pathogenesis, in which our interest has recently been aroused by the stimulating suggestions of Rosenow and Billings. A decade ago the prominent subjects in thyroid literature were early diagnosis, obscure points in diagnosis, formes frustes, etc., yet in spite of the thoroughness with which the question of diagnosis has been exhausted, there is apparently a considerable number of fairly well-marked cases of hyperthyroidism in which the diagnosis is not made, or is greatly obscured by the undue prominence of certain of the less common symptoms. The cases here reported may serve to point out the possibility of such diagnostic error, and to emphasize again the necessity of keeping the thyroid in mind in considering many rather obscure clinical pictures.

Case 1.—Miss F., student of 21 with unimportant family and past history, consulted her family physician in January, 1915, complaining of loss of appetite and malaise. She was found to have a temperature of 103.4; white blood count 9600; Widal negative. Urotropin was given. After a few days began to have frequent burning urination, and blood was discovered in the urine. Temperature rose to 100-103 every afternoon. Tuberculosis of the urinary tract being suspected, the urine was sent to a laboratory for guinea-pig inoculation, and four weeks later the laboratory reported positive tuberculosis. A diagnosis of tuberculosis of the kidney was made, but on account of the absence of any indication of tuberculosis in the ureteral urines, the patient was brought to the hospital in May, 1915, for further investigation.

Physical examination showed a small, well-developed young woman. Thyroid moderately prominent. Marked vasomotor flushing about chest. Pulse 100-124. Systolic blowing murmur at cardiac apex. Slight general abdominal tenderness. Tremor of hands. Knee-jerks lively. Urine 1.011 with trace of albumin and rare hyaline cast. White blood cells 8500: polymorphonuclear 57%, lymphocytes 40%. Hemoglobin 70%. Afternoon temperature 99 to 99.5. Cystoscopic examination showed mild cystitis and some things suggestive of pyelitis. Guinea-pig inoculations negative.

In this case, the rather striking fever at onset naturally occupied the attention of the attending physician, and the attempts to explain it on the basis of kidney infection and to influence that infection by means of hexamethylenamine totally obscured the picture of hyperthyroidism that was doubtless developing during the weeks before the patient came to the hospital. With prolonged rest, overfeeding, hydrobromide of quinine and a discontinuance of bladder therapy there was practically complete relief from symptoms.

Case 2.—Miss W., schoolteacher, complaining of nervousness. Has had five attacks of pneumonia, and has had occasional periods of loss in weight, nervousness and irregular menstruation, but has been able to continue her work. Past history otherwise unimportant. In the summer of 1914 she began to be troubled with nervousness, cardiac palpitation and marked tremor, and lost ten pounds in weight. Physical examination at this time showed slight prominence of eyes, slight enlargement of thyroid; heart rate 120 with systolic blow at apex. A diagnosis of hyperthyroidism was made, and with prolonged rest, quinine hydrobromide, iodine ointment applied over the gland, and a copious non-irritating diet, improvement was marked.

Patient remained practically well up to the middle of January, 1915, when she had an apparent influenza infection for several days, with considerable fever, cough, and some rales in right chest. Was in bed two weeks, the fever continuing, at times as high as 102.5. Pulse 110-120. At the end of this time the temperature fell to normal, the pulse remaining 90 to 100. After a week or so the temperature again rose, and for a period of three weeks reached 100 to 101 each evening. A marked increase in the size of the thyroid was noted with the second rise in temperature. She continued to lose weight, had some sweats, considerable tremor and rapid heart action. White blood count 3700: polymorphonuclears 56%, lymphocytes 36%. Urine normal. X-ray of chest showed nothing positive. Following x-ray treatment of the thyroid, the gland was reduced to about half its former size and became firm, with well-defined borders. Leucopaenia throughout. Widal negative. With continued rest in bed, iodine ointment and quinine hydrobromide in addition to the x-ray therapy, there was gradual improvement. Temperature fell to normal after about three weeks, and remained so except for an occasional transient rise to 99 or 100. Pulse 90 to 110 throughout. Patient left the hospital after four months, weighing more than ever before. No sweats. Eyes practically normal. Thyroid firm and small. No heart murmur. Very little tremor. Has remained well to the present time.

Here, again, without a definite knowledge of the previous attacks, one might well be misled by the striking temperature chart; and of course at the onset of such an attack as the last one the diagnosis must be held in abeyance until all the more usual causes of fever can be excluded.

The question of the frequency and extent of temperature elevation in exophthalmic goiter is one in which there is still difference of opinion. Bertoye, who in 1888 first made a detailed study of the matter, concluded from an analysis of a considerable number of cases that moderate transient fever is of frequent occurrence, and may be found at the onset, during the course of the disease, or only terminally. Kocher, on the other hand, does not consider that fever is a part of the picture in exophthalmic goiter at all, and agrees with Mackenzie that a temperature over 100 is exceedingly rare. In this country, casual mention is made by Barker, and in the papers from the Mayo clinic, of occasional slight fever during the course of the disease, but no emphasis has been laid on its occurrence except by W. Gilman

* Read before the annual meeting of the California State Medical Society, Fresno, Cal., April 20th, 1916.